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cal approach at this time is to withhold all drugs of any description during the first trimester of pregnancy, unless their administration is absolutely indicated.

The authors would like to lend their support to the proposals in a Leading Article in the February 10, 1962 issue of the *Lancet*[8] wherein it is suggested that lists be kept of all drugs given to pregnant women during the first trimester and that these lists be examined critically whenever structural or biochemical abnormalities of the fetus or newborn occur. Furthermore, we would strongly advocate a national or international centre to which such data could be sent by various physicians, for compilation and comparison. This would surely represent an opportunity to apply the non-human electronic computers to a very humane project.

SUMMARY

A case of phocomelia occurring in the offspring of a mother who received thalidomide (Kevadon) in the first trimester of pregnancy is reported. Although the association in this case is suspected only, the implications are far-reaching for both present and future generations. A plea is made for the establishment of centres for assimilation of data relating to maternal drug therapy and congenital anomalies.

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Gilles de la Tourette Syndrome Treated by Bimedial Frontal Leucotomy

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GILLES de la Tourette syndrome is an involuntary paroxysmal hyperkinesis involving the entire skeletal musculature. It is characterized by onset in childhood, a diagnostic triad, and a progressive course throughout adult life toward personality and motor disorganization. The diagnostic triad consists of (1) explosive, stereotyped somatomotor tics; (2) abrupt repetitive vocalizations which typically progress to forced shouting of obscenities (coprolalia); and (3) forced mimicry of the talk and actions of others (echolalia and echopraxia). There is usually an associated personality disorder. The syndrome must be differentiated from chorea, postencephalitic parkinsonism, and hysterical and schizophrenic mannerisms.

There are occasional reports of remission in association with a variety of attempted treatment measures. Michael[1] reported an 18-month remission attributed to treatment by carbon dioxide narcosis. Eisenberg, Ascher and Kanner[2] described a well-documented remission that was considered to be due to intensive family psychotherapy. Treatment is usually ineffectual, however, and spontaneous remissions are exceedingly rare. An excellent discussion of the subject has been provided by Mazur.[3]

The cause of this disorder is unknown. Autopsy findings[2] are equivocal or entirely normal. Typically, there is no clinical evidence of central nervous system involvement. Motivational (psychodynamic) explanations invoke hostility-mockery responses to one or both parents.

The patient described in this report showed more evidence suggestive of birth trauma than do most cases of this syndrome. As far as we can ascertain, this is the first case of Gilles de la Tourette syndrome to undergo leucotomy.

R.M., an unemployed single man, aged 22 years, presented in the summer of 1960 to the psychiatric department of Toronto Western Hospital. He complained of somatomotor and vocal tics since the age of 9 years; severe panic attacks with depersonalization since 20; and increasing feelings of hopelessness.

In addition there were longstanding complaints of genital and rectal autostimulation, sadistic "torture" fantasies referable to adult females, and "hatred" of his father.

His birth history describes a traumatic delivery after a three-day trial of labour. A second sibling who is normal was delivered by Cesarean section. At birth this patient was hypotonic and unable to cry or suckle, and the left eyelid twitched. The head was "markedly" deformed. He was force-fed.

Motor development was slow with marked weakness and incoordination. Speech commenced at 30 months but was precocious thereafter. He exhibited the restlessness, irritability and distractibility which are usually associated with brain damage from birth. A grand mal seizure was recorded at the age of three years and again at the age of five.

Motor and vocal tics commenced simultaneously at age nine and gradually worsened through the intervening years. The patient was enuretic until the age of 12 years. At age 16 (grade IX) he was released from

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school because of his tics—in spite of fair scholastic achievement. He underwent surgical correction of a right esotropia at age 16 with a good result.

Neuropsychiatric treatment since childhood, including one admission to mental hospital, was of no avail. Chlorpromazine in doses of 600-700 mg./day afforded a slight reduction of the tic, but did not affect the panic or other symptoms.

The patient was a tall, thin, intelligent young man whose basic poise was constantly interrupted by sudden jerking movements of the axial skeleton and limbs accompanied by short shouts. One of his many tics resembled an elaboration of spasmodic torticollis with the additional feature of cupping the ipsilateral ear with one hand and shouting "eh!" The patient was usually able to suppress coprolalic ejaculations in company—but not always.

His mental status, apart from the manifest syndrome and the mushrooming effect of social rejection and treatment failure, appeared normal. His functioning I.Q. was 110. Personality tests, as in the past, suggested obsessional personality with immature and/or latent schizophrenic features.

There was longstanding temporal pallor of the right optic disc, with an associated right nasal upper quadrant visual field defect. There was hypertrophy of the left sternomastoid muscle in keeping with the torticollic component. Physical examination was otherwise normal, as were the cerebrospinal fluid, electroencephalogram, air encephalogram and right carotid arteriogram.

The patient was admitted to hospital, chlorpromazine treatment was withdrawn, and a period of observation was instituted. He consistently twitched and emitted involuntary shouts throughout his waking hours. The frequency and severity of the tics were seen to parallel the degree of emotional tension. For example, an explosive tic occurred with high frequency (14 times/minute) throughout a half-hour presentation to a class of student nurses.

On October 19, 1960, a bimedial leucotomy was performed. An unfortunate postoperative complication of staphylococcal frontal lobe abscess with grand mal seizures responded to aspiration of the abscess, antibiotics and anticonvulsants.

Following the operation, his tics and panic attacks were markedly reduced. When the patient was again presented to the student nurses two months postoperatively, only two minor tics could be observed in a half-hour period.

The patient was discharged on a regimen of 600-800 mg. chlorpromazine daily, plus continued diphenylhydantoin (Dilantin). (A preoperative trial of diphenylhydantoin had had no effect on the syndrome.)

The remission has been sustained to date (December 1961), one year after discharge from hospital. The patient is attending night school regularly. His tics and panics are minimal. He no longer occupies his time with destructive fantasies and now has hope of a reasonable future. He still exhibits a moderately short attention span and a dearth of ordinary social contacts. His postoperative I.Q. is 115.

DISCUSSION

Gilles de la Tourette syndrome is due either to an organic lesion or to faulty learning or both. If it is due to a lesion, it may perhaps be regarded as an unusual form of cerebral palsy. With regard to the lesion and learning combination, it is conceivable that the lesion could involve the learning-recording mechanism of the brain and brainstem in such a way that faulty learning is created no matter how bland the environment.

If the learning mechanism is normal and if there is no motor lesion, one must postulate a highly abnormal input, e.g. traumatic child-parent relationships.

Foltz, Knopp and Ward[4] produced experimental spasmodic torticollis in monkeys by placing a small lesion in the medial reticular substance of the tegmentum of the mid-brain. The experimental torticollis reproduced all of the features of human spasmodic torticollis, including marked accentuation by emotional stress. I venture to suggest that some such lesion may underlie the Gilles de la Tourette syndrome, and that electron microscopy might reveal such a lesion.

The decision to perform leucotomy in this case hinged on the usual issue: how much reduction of tension could be expected, at the expense of how much loss of "control", by cutting the frontothalamic radiations bilaterally? There was clear evidence of disabling panic attacks which had not responded to treatment. What chance was there that the sadistic fantasies or the "hopelessness" would be affected by leucotomy? The tics were considered to represent the result of an erratic balance between impulse expression and impulse control, whatever the etiology might be. Which way would leucotomy affect this balance? It was decided to subject this patient to leucotomy with a view to reducing his attacks of panic, hoping also to benefit the tic.

One year after operation, improvement is maintained in all of his symptoms. The patient is "socially acceptable". It is, of course, too early to make a final assessment of the value of the treatment in this case.

SUMMARY

A case of Gilles de la Tourette syndrome is reported. The patient has maintained remission for one year following bimedial frontal leucotomy. Diagnostic, etiologic and therapeutic issues are discussed.

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